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comorbid clinical symptoms and disorders. Transition rate from CHR to full psychosis in untreated cohorts is not known. In some patients, persistent comorbid symptoms could indicate a prolonged prodromal phase and increased risk of late transition. Although treatment in CHR patients focuses mainly on interventions aiming to decrease the risk of transition, comorbid symptoms and disorders, which often tend to persist over a long period and contribute to psychosocial impairment, must not be neglected in the monitoring and treatment of CHR patients. Future research could also identify potential critical intervention points where success may alter the life course of illness.

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Psychosis as a side effect of COVID-19 vaccine administration – a case series

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Background: The COVID-19 pandemic has had a major impact on people's mental health, being a significant psychological stressor that has precipitated anxiety and stress-related disorders [1], [2]. However, it is being studied how the virus itself can generate neuropsychiatric symptoms such as psychotic or confusional ones [1], [2], [3]. The pathogenic mechanisms underlying the psychiatric manifestations are not clear but there are different theories: direct invasion of the central nervous system as occurred in previous coronavirus infections, inflammatory response generated in the central nervous system with the release of large amounts of cytokines, peripheral immune cell transmigration into the central nervous system and post-infectious autoimmunity [1], [2]. Regarding the causal relationship between COVID-19 and psychosis, numerous confounding factors such as lack of clinically relevant details, including whether and how delirium was excluded, or overreporting of cases are described. At any rate, in the scientific literature we can find a number of case reports linking new onset psychotic symptoms to COVID-19 [3], although there are very few that relate these symptoms to COVID-19 vaccines [4], [5].

Objective: The aim of the study is to investigate whether there is a causal relationship between the administration of the COVID-19 vaccine and the onset of psychotic symptoms in patients without a previous mental health history.

Methods: A case series of two patients with first psychotic episode after administration of this vaccine is presented. Both patients were attended by the psychiatric unit of the Regional University Hospital in Malaga.

Results: Two male patients, 23 and 30 years old, with no previous history of mental illness or substance use presented with auditory and cenesthetic hallucinations, disorganized behavior and speech, and a feeling of derealization 24-72 hours after COVID-19 vaccine administration. On the first day they had presented febrile fever and fatigue as somatic symptoms. One of them also had insomnia and delusions, with significant psychotic distress, which required hospitalization in the psychiatric ward. Physical examination and complementary tests were normal. In both patients, with the introduction of low antipsychotic doses, the symptomatology resolved almost completely within a week, with good insight.

Conclusions: There are limited data on the side effects of COVID-19 vaccines and very few case reports have described an onset of psychosis following administration of this vaccine.

Studies have shown that SARS CoV-2 can trigger an immune response with the release of cytokines that could explain neuropsychiatric symptoms. On the other hand, the vaccine would trigger a similar, although more attenuated, immune response that could also be responsible for the onset of psychotic symptoms observed in these two patients. Thus, it is hypothesized that psychosis may be related to a fast increase in the proinflammatory response and an activated autoimmune state either post-infectious or post-vaccination.

To conclude, although there are confounding factors and further research would be needed to clarify a causal relationship, we believe that longitudinal monitoring of the neuropsychiatric symptoms in those patients exposed to the COVID-19 vaccine would be of interest.

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Pro-inflammatory systemic state in schizophrenia is associated with an increased intestinal inflammation and permeability

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Introduction: The relationship between systemic inflammation and schizophrenia (SZ) has been widely described (1). C-reactive protein (CRP) is a commonly used unspecified biomarker of systemic inflammation worldwide, and meta-analysis of Fernandes et al. [2] found increased level of CRP in SZ patients. Intestinal bacterial translocation has been postulated as one of the causes of systemic inflammation [3, 4, 5].

Objectives: 1) To describe the prevalence of pro-inflammatory systemic state in patients with schizophrenia, based on C-reactive protein blood levels. 2) To analyze factors related to pro-inflammatory systemic state in these patients, including indirect biomarkers of intestinal bacterial translocation.

Methods: Cross-sectional study of 80 patients with schizophrenia [mean age=42.08 (SD=12.46; range 20-67); males=56 (57.5%)], recruited from their mental health outpatient clinics in Oviedo (Spain). Assessment: a) Psychometric assessment: PANSS, Clinical Assessment Interview of Negative Symptoms (CAINS), Calgary Depression Scale (CDS). b) Pro-inflammatory state: C-reactive protein (CRP) (mg/L), and indirect markers of intestinal bacterial translocation (lipopolysaccharides binding protein -LBP-, soluble CD14 -sCD14-) in plasma. Metabolic syndrome -MetS- [ATP III criteria: based on glucose, HDL, triglycerides (mg/dl), arterial pressure (AP) (mmHg), abdominal circumference (cm)], body mass index (BMI) (kg/cm²), adherence to Mediterranean Diet, physical activity (IPAQ scale), smoking, cannabis or alcohol use and chlorpromazine equivalent doses were recorded. Statistical analyses: Chi-square, t-Student test, logistic regression analyses (Forward stepwise).

Results: Patients were mostly clinically stable, presenting slightly higher negative symptom score and lower positive and depressive symptom scores, and had a mean length of illness of 14.11 (SD=11.19) years. Most patients (93.8%; n=75) were on antipsychotic maintenance treatment. Mean BMI was 29.22 (SD=5.49) and prevalence of MetS was 43.8% (n=35). Prevalence of tobacco smoking was 35%, but only 6 patients (7.5%) had used cannabis last month.

Systemic inflammation (SI) was defined as blood levels of CRP >0.3 mg/dl, being present in 41.3% of patients [mean CRP levels 0.64 mg/dl (SD=1.19)].

Patients with CRP >0.3 mg/dl (group with SI) did not differ from the non-inflammatory group in age, sex, length of illness, diet or physical activity, tobacco, alcohol or cannabis use, benzodiazepine or antidepressant use, chlorpromazine equivalent doses, nor presence of MetS. However, this group had higher BMI (31.07±6.21 vs 27.92±4.56 kg/m², t=-2.616, p=0.011), and increased levels of LBP (17.17±5.27 vs 11.65±3.29, t=3.757, p<0.001) and sCD14 (2.16±0.43 vs 1.82±0.36, t=2.481, p<0.001). LBP and sCD14 were significantly correlated (r=0.400, p<0.001). Regarding clinical severity scores, patients with SI had lower scores on PANSS-P (10.61±4.0 vs 12.81±4.88, t=-2.136, p=0.036), but no statistically significant differences were detected in other clinical scores.

Logistic regression analyses including independent factors (age, sex, PANSS-P, BMI, LBP and sCD14) showed that patients with SI had increased plasma levels of LBP (OR 95%CI: 1.277 – 2.078, p<0.001), and lower PANSS-P (OR 95%CI: 0.723 – 0.983, p=0.030).